Supplemental Fig 1. RHCE genotyping assays. Cc and Ee genotyping assays are based on (i) PCR co-amplification of *RHD* and *RHCE* regions using dye-labelled primers, (ii) digestion of the PCR products with *Mnl*I with digestion pattern dependant on the *RHCE* allelic status, and (iii) quantitative comparison of the peak areas using an Applied Biosystems 3130xl Genetic Analyzer and GeneScan v3.0 software, taking into account knowledge of *RHD* copy number. To ensure complete digestion internal control restriction enzyme sites were included in each assay: complete digestion was confirmed by the absence of a peak at the original product size (179bp for the Cc assay and 177bp for the Ee assay). Several polymorphisms contribute to the RHC antigen; the only one showing perfect correlation with antigenicity is the P103S polymorphism (rs676785; proline is c, serine is C; Avent & Reid 2000 *Blood* 95:375-87). Amplification across this site is achieved with the primers L: 5′-TTCTGGAACCTGTCCTTTCG-3′ and R: 5′-FAM-GTGTGGCCTTCAAGCTCTTC-3′. *MnlI* digestion occurs in the presence of the c allele (88 bp) but not the C allele (138 bp; the *RHD* gene copies also are not digested). RHE genotyping relies on amplification across the variable site rs609320 (A226P, with the alanine representing e, proline representing E) with the primers L: 5′-HEX-TGCTCACCWTGCTGATCTTC-3′ and R: 5′-CAGGCGCCCTCTTCTTGT-3′. *Mnl*I digestion occurs in the presence of the E allele (122 bp) but not the e allele (159 bp; the *RHD* gene copies are also not digested). Both assays were performed in a duplex PCR and digestion. Seven positive control DNA samples (six of which were from individuals with known Rh serotype) were run with each plate. Example results representing different *RHD* copy number states are shown below.